TOBACCO SMOKE COMPONENTS

Tobacco smoke is a complex mixture of thousands of components. Over the years, much time and effort has been devoted simply to determining the identity of those components. This has proven difficult, however, and much remains to be learned about the structure and makeup of tobacco smoke.

Despite these problems, tobacco smoke or one of its components (usually "tar," nicotine or carbon monoxide) is commonly assumed to cause disease. Such assumptions are based primarily on laboratory experiments using extremely large amounts of these substances -- quantities which may seriously affect the experiments' relevance to humans. But neither "epidemiological studies, nor animal and clinical studies, have identified any ingredient or group of ingredients as found in smoke as disease producing in humans."[1] [Emphasis added]

"Tar"

The frequent use of the word "tar" has created the impression that it is present in tobacco smoke. But this is not correct. The substance called "tar" is actually a laboratory product obtained by collecting the

particulate matter in tobacco smoke. The collection method most often used involves passing cigarette smoke through a cold trap at extremely low temperatures. Material collected in this way hardly duplicates what humans are exposed to in the smoking process.

Even if the "tar" obtained by these methods were exactly the same as the smoke inhaled by the smoker, it still might be very different by the time it is studied in the laboratory. That is because, once collected, it continues to undergo chemical changes, even while stored on the laboratory shelf.[2]

Despite its probable lack of relevance to the smoking process, "tar" has been used in animal experiments studying the relationship between tobacco smoke and cancer. In many of those experiments, "tar" was repeatedly painted on the skin of test animals over prolonged periods of time. Any tumors that developed were then compared with lung tumors in humans.

For obvious practical as well as technical reasons, it is apparent that what was done to those laboratory animals is not what happens in the human body. After all, how can skin painting be compared to tobacco smoke inhalation, and how can the skin of animals be compared to the cells which line the human lung?

Many scientists have been dubious about making such comparisons for years, warning about the difficulties that must be considered in projecting the results of animal experiments to the human situation. One scientist went so far as to say that such experimental evidence was "claptrap."[3]

Another major difficulty involved in studying "tar" centers around the difference between quality and quantity. An experimental toxicologist said in 1976 that the effect in a particular species does not necessarily mean that it might "occur either quantitatively or qualitatively in man."[4] He also stated:

> "Agents have been identified in 'tar' which might in theory have a carcinogenic effect, but the qualitative identification of a particular agent in a complex mixture does not mean that it is present in sufficient quantities to produce a biological effect. . . ."[5]

All in all, the current state of scientific evidence concerning "tar" was concisely summarized during a Congressional hearing:

> "Human beings do not smoke 'tar' and no significance of laboratory reports on 'tar' yield to human health has been established."[6]

Nicotine

Although nicotine is present in tobacco smoke, it is rapidly absorbed and excreted. Consequently, nicotine has been described as having no known chronic or cumulative effects. Still, nicotine has been blamed for playing a role in the development of heart disease.

In examining that assertion, two European scientists who have been involved in this area discussed nicotine and disease causation. When asked about a possible effect of nicotine on the blood vessels, a researcher who is generally antismoking replied that such an effect is "quite harmless" and "we therefore do not believe that nicotine can cause arteriosclerosis."[7] The other scientist, who based his response on his "long years of experience," is "convinced that nicotine does not damage the healthy heart." He stated "there are no indications that nicotine plays a part in the causation of chronic cardiovascular disease . . ."[8]

Another discussion, between two American scientists invited to attend a one-day conference on cigarette smoke components, reflects similar conclusions. When asked "whether nicotine is related to cardiovascular disease and whether coronary disease or atherosclerosis is related to nicotine," a physician with expertise in this area replied, "There are no data on that."[9]

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Like the "tar" experiments, animal studies on nicotine and heart disease have been roundly criticized for their unrealistic and excessive test conditions:

> "There have been some studies which have exhibited minor or questionable changes with the use of an equivalent dose of 600 or more cigarettes a day in man. This is such a large number that I think man would find it difficult to find the time to smoke them."[10]

The relevance of their results also must be questioned, because other animal experiments using more realistic doses of nicotine have failed to show any influence on the atherogenic process:

> "In these experiments it was clearly demonstrated in the rabbit that realistic doses of either nicotine or cigarette smoke failed to initiate, exacerbate, or otherwise influence the atherogenic process in that species."[11]

A particularly telling comment about the alleged role of nicotine in disease causation is provided by a recent report on the status of a federal smoking and health program. Although much of the report is predictably anti-smoking, it concedes that "evidence linking it [nicotine] to the chronic pathogenic effects of smoke is not available."[12]

In summary: "While many studies have been done in this field, none have established nicotine as contributing to the causation, aggravation or precipitation of any cardiovascular disease."[13]

Carbon Monoxide

Carbon monoxide (CO) is a tasteless, odorless gas produced by many natural and man-made sources, including automobile exhaust fumes and industrial emissions. Burning cigarettes also produce carbon monoxide but that amount has been described as "insignificant" compared to those other sources.[14] It is also naturally produced during body metabolism.

To support their health claims about the adverse effects of CO, anti-smoking advocates have emphasized the results of certain animal studies, particularly those published by a team of Danish scientists who exposed rabbits to large quantities of CO and a high cholesterol diet. The exposed rabbits reportedly developed vascular changes similar to early atherosclerosis in man.[15]

In 1977, a prominent health investigator apparently was thinking about those experiments when he answered his own rhetorical question, "Does CO play a role in arteriosclerosis?" Although he is definitely a member of the anti-smoking establishment, he admitted that "it certainly works in rabbits, but there's considerable doubt whether it works in man."[16]

Recently, those same Danish researchers announced that it may not work in rabbits, either. They had decided to repeat their earlier tests after learning that their results had not been duplicated in experiments using monkeys, baboons, dogs, and pigeons.[17] They were unable to duplicate their own results:

> "Investigation revealed that irrespective of duration or level of exposure [to CO], no significant morphological changes were present to discriminate between experimental and control animals."[18]

In an apparent reference to that development, a German researcher reflected on its potential impact:

> "Now as regards the role of carbon monoxide, some two years ago we believed that in CO, we had found the evil doer. Unfortunately, it turned out that the tests in question had not been interpreted quite correctly so that today we have to back off from the CO hypothesis as far as the causation of arteriosclerosis is concerned."[19]

What can one conclude from all of this discussion? At best, one can say that many questions remain unanswered about the role of tobacco smoke and its components in disease causation. In the words of one researcher who has tried

to establish a causal link between smoking and disease:

> "We assume that it is the tar which causes lung cancer, but we do not know this for certain.
>
> Until now, we have implicated nicotine in the development of cardiovascular diseases, but we cannot prove this.
>
> The same is true for CO."[20]

Obviously, such indecisive words from an anti-smoker demonstrate that statements asserting cause-and-effect are at best oversimplifications of the scientific data.

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